PCT WORLD INTELLECTUAL PROPERTY ORGANIZATION
International Bureau

INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)

(51) International Patent Classification 6 : A61M 1/10, 25/10

A1

(11) International Publication Number: **WO 99/51286**

(43) International Publication Date: 14 October 1999 (14.10.99)

(21) International Application Number: PCT/US99/07411

(22) International Filing Date: 2 April 1999 (02.04.99)

(30) Priority Data:
09/054,214 2 April 1998 (02.04.98) US

(71) Applicant: SCIMED LIFE SYSTEMS, INC. [US/US]; One Scimed Place, Maple Grove, MN 55311–1566 (US).

(72) Inventor: TALPADE, Dnyanesh; 12105–41st Avenue North #331, Plymouth, MN 55441 (US).

(74) Agents: KOEHLER, Steven, M.; Westman, Champlin & Kelly, P.A., International Centre, Suite 1600, 900 Second Avenue South, Minneapolis, MN 55402–3319 (US) et al.

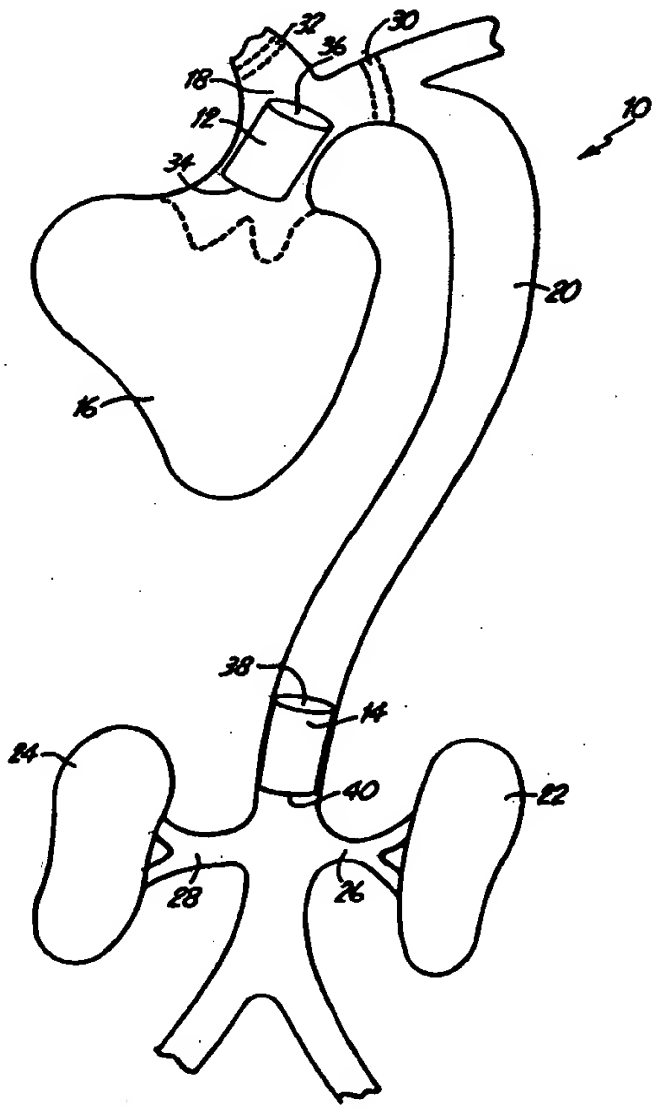
(81) Designated States: CA, JP, European patent (AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE).

Published
With international search report.
Before the expiration of the time limit for amending the claims and to be republished in the event of the receipt of amendments.

(54) Title: SYSTEM FOR TREATING CONGESTIVE HEART FAILURE

(57) Abstract

A system (10) is provided for regulating blood flow to a portion of the vasculature, such as the renal system, in order to treat heart disease. A regulator (12, 94') maintains blood flow so as to control physiological feedback responses in order to relieve overload conditions on the heart.



FOR THE PURPOSES OF INFORMATION ONLY

Codes used to identify States party to the PCT on the front pages of pamphlets publishing international applications under the PCT.

AL	Albania	ES	Spain	LS	Lesotho	SI	Slovenia
AM	Armenia	FI	Finland	LT	Lithuania	SK	Slovakia
AT	Austria	FR	France	LU	Luxembourg	SN	Senegal
AU	Australia	GA	Gabon	LV	Latvia	SZ	Swaziland
AZ	Azerbaijan	GB	United Kingdom	MC	Monaco	TD	Chad
BA	Bosnia and Herzegovina	GE	Georgia	MD	Republic of Moldova	TG	Togo
BB	Barbados	GH	Ghana	MG	Madagascar	TJ	Tajikistan
BE	Belgium	GN	Guinea	MK	The former Yugoslav Republic of Macedonia	TM	Turkmenistan
BF	Burkina Faso	GR	Greece			TR	Turkey
BG	Bulgaria	HU	Hungary	ML	Mali	TT	Trinidad and Tobago
BJ	Benin	IE	Ireland	MN	Mongolia	UA	Ukraine
BR	Brazil	IL	Israel	MR	Mauritania	UG	Uganda
BY	Belarus	IS	Iceland	MW	Malawi	US	United States of America
CA	Canada	IT	Italy	MX	Mexico	UZ	Uzbekistan
CF	Central African Republic	JP	Japan	NE	Niger	VN	Viet Nam
CG	Congo	KE	Kenya	NL	Netherlands	YU	Yugoslavia
CH	Switzerland	KG	Kyrgyzstan	NO	Norway	ZW	Zimbabwe
CI	Côte d'Ivoire	KP	Democratic People's Republic of Korea	NZ	New Zealand		
CM	Cameroon			PL	Poland		
CN	China	KR	Republic of Korea	PT	Portugal		
CU	Cuba	KZ	Kazakstan	RO	Romania		
CZ	Czech Republic	LC	Saint Lucia	RU	Russian Federation		
DE	Germany	LI	Liechtenstein	SD	Sudan		
DK	Denmark	LK	Sri Lanka	SE	Sweden		
EE	Estonia	LR	Liberia	SG	Singapore		

SYSTEM FOR TREATING CONGESTIVE HEART FAILURE

BACKGROUND OF THE INVENTION

The present invention deals with treatment of heart disease. More particularly, the present invention deals with a system and method for treating heart disease by regulating blood flow in the vasculature.

Congestive heart failure is a common heart disease. The prevalence of incidents of congestive heart failure has recently increased, and there is considerable morbidity and mortality associated with its diagnosis. In fact, congestive heart failure is an extremely lethal disease with an estimated five year mortality for a vast majority of both men and women who encounter the disease.

Congestive heart failure results from loss of, or impairment of, normal heart function. This loss or impairment reduces cardiac output. This, in turn, results in a reduction in both blood flow and blood pressure in the kidneys. This reduction in flow and pressure causes a renin-angiotensin response that exacerbates congestive heart failure.

Briefly, as blood flow and pressure is reduced in the kidneys, cells in the kidneys referred to as juxtaglomerular apparatus secret an enzyme referred to as renin into the blood. The enzyme renin cleaves a ten-amino acid polypeptide called angiotensin I from a plasma protein in the blood called angiotensinogen. A converting enzyme in the blood removes two amino acids from the angiotensin I polypeptide leaving an eight amino acid polypeptide called angiotensin II. Angiotensin II has numerous effects on the smooth muscle layers of arterioles, including causing

vasoconstriction. Further, an indirect effect of an increase in angiotensin II increases blood volume. Blood volume is increased because angiotensin II stimulates secretion of aldosterone from the adrenal cortex which, in turn, causes an increase in salt and water retention in the kidneys. Angiotensin II also stimulates thirst centers in the hypothalamus causing more water to be ingested. The increase in blood volume and the corresponding vasoconstriction cause an increase in blood pressure and hence a volume overload on the heart which causes further deterioration of the heart condition.

Another response is also related to congestive heart failure. Baroreceptors, referred to as stretch receptors, reside in the aortic arch and carotid sinuses. The baroreceptors are essentially pressure sensors sensing blood pressure in that area. The baroreceptors provide physiological feedback in two ways. First, in response to a reduction in blood pressure, the baroreceptors provide a neurohormonal feedback response which acts to increase the heart rate in an attempt to increase cardiac output. The increased heart rate causes the heart to work harder which, in turn, causes the heart muscle to stretch further. Also, a reduction in pressure caused by a reduction in cardiac output causes the baroreceptors to provide a feedback response which acts to constrict the distal vasculature thus increasing pressure in that area.

It can thus be seen that impairment of heart function can lead to a cyclical feedback response which increases, rather than reduces, the impairment. Such a cyclical feedback response is sometimes referred to as a cascade.

For instance, if the heart muscle is stressed, the heart works harder and begins to stretch. This reduces the efficiency of the heart. This inefficient or impaired heart function causes blood pressure in the areas of both the kidneys and the baroreceptors to decrease. The feedback response generated by the kidneys causes further overload and stress on the heart. The feedback response generated by the baroreceptors causes increased heart rate. Both of these feedback responses cause the heart to work harder, causing further stretching of the heart muscle and thus leading to greater inefficiencies. In response, the feedback responses become even more acute -- and the cascade continues.

SUMMARY OF THE INVENTION

A system is provided for regulating blood flow to a portion of the vasculature, such as the renal system, in order to treat heart disease. A regulator maintains blood flow so as to control physiological feedback responses in order to relieve overload conditions on the heart.

In one embodiment, a system is provided for treating heart disease in a mammal having a heart, an ascending aorta, a descending aorta, and a renal system including renal arteries. The system includes a first disposed in the ascending aorta and having an inflow end and an outflow end. The first regulator receives blood flow at a first velocity of the inflow end and provides blood flow at a second velocity through the outflow end thereof, wherein the second velocity is lower than the first velocity. A second regulator is disposed in the ascending aorta upstream of the renal arteries. The second regulator has an inflow end and an outflow end and receives blood flow at a third velocity at the

inflow end and provides blood flow at a fourth velocity through the outflow end thereof. The fourth velocity is greater than the third velocity.

In a second embodiment, a plurality of expandable members are placed across the renal arteries and/or baroreceptors to maintain blood flow and pressure to the renal arteries and/or baroreceptors and to thus inhibit undesirable responses from the renin-angiotensin system.

BRIEF DESCRIPTION OF THE DRAWINGS

FIG. 1 illustrates a system for treating heart disease in accordance with one aspect of the present invention.

FIG. 2 is a top plan view of a pump in accordance with one aspect of the present invention.

FIG. 3 is a side view of a portion of the pump shown in FIG. 2.

FIG. 4 is a side view of a portion of the pump shown in FIG. 3.

FIG. 5 is an opposite side view of a portion of the pump shown in FIG. 3.

FIG. 6 is an end view looking into the pump illustrated in FIG. 2.

FIGS. 7A and 7B illustrate deployment of a blood flow regulating system in accordance with one aspect of the present invention.

FIG. 8 is a plot of sinus rhythm against balloon inflations and deflations in accordance with one aspect of the present invention.

FIGS. 9A-9D illustrate operation of the system shown in FIG. 7.

FIG. 10 illustrates a catheter implementing a portion of the blood flow regulating system shown in FIG. 7.

DETAILED DESCRIPTION OF THE PREFERRED EMBODIMENTS

FIG. 1 illustrates a flow regulating system 10 in accordance with one aspect of the present invention. Flow regulating system 10 includes a first flow regulator 12 and a second flow regulator 14. Flow regulators 12 and 14 are placed in the vascular system of the patient, wherein the vascular system includes heart 16, ascending aorta 18, descending aorta 20, kidneys 22 and 24, and renal arteries 26 and 28. The vascular system also includes baroreceptors 30 and 32 located proximate ascending aorta 18.

Flow regulator 12 has an inflow end 34 and an outflow end 36. Blood from heart 16 flows into regulator 12 through inflow end 34 and flows out of regulator 12 through outflow end 36. The blood then travels through ascending aorta to the rest of the vasculature, including across baroreceptors 30 and 32, down descending aorta 20 and into flow regulator 14. The blood flows out of regulator 14 to the renal arteries 26 and 28.

In one preferred embodiment, flow regulator 12 regulates the velocity of blood flowing from heart 16 to the ascending aorta 18 by decreasing the velocity of the blood flow from a first velocity entering inflow end 34 to a lower velocity exiting outflow end 36. Regulator 14 also has an inflow end 38 and an outflow end 40. Regulator 14 regulates the velocity of blood flowing in the inflow end 38 and increases the velocity to a higher level as it flows through outflow end 40.

By reducing the velocity of blood flow from the inflow end 34 of regulator 12, through its outflow end 36, the velocity of blood flow encountered by baroreceptors 30 and 32 is lower as well. This induces a classic sympathetic nervous response. In other words,

the blood pressure encountered by baroreceptors 30 and 32 will be reduced. This causes baroreceptor 30 and 32 to generate a neurohormonal response which calls for heart 16 to beat at an elevated rate and which induces vasoconstriction. This increases the mean arterial pressure in the vasculature.

However, since flow regulator 14 acts to increase the blood flow velocity therethrough, which acts to increase the blood pressure in renal arteries 26 and 28. This, in turn, inhibits the classic renal response to hypotension thus inhibiting the vascorestriction and increased blood volume associated with that response. This reduces the overload and stress on heart 16.

The overall reduction in stress on heart 16 allows passive rehabilitation of the myocardial system (i.e., the heart can repair itself). This also increases the efficiency of pharmacologically supplemented rehabilitation of the myocardium.

Flow regulators 12 and 14, in one preferred embodiment, are of similar construction. Flow regulator 12 acts as a step up regulator and flow regulator 14 acts as a step down regulator. However, as will be discussed later in the specification, regulators 12 and 14 can be substantially identical regulators, and can simply be positioned in the vascular system in 180° opposing relation to accomplish the desired step up or step down function.

FIG. 2 is a top plan view, taken in partial section, of flow regulator 12. It will be appreciated that flow regulator 14 is substantially identical to flow regulator 12, and therefore only flow regulator 12 will be described in detail. FIG. 2 illustrates that flow regulator 12 preferably includes a housing 42 which

houses a pair of centrifugal pumping mechanisms 44 and 46. Also, housing 42 has a pair of walls 48 and 50 which, in combination with pumping members 44 and 46, act to separate housing 42 into two chambers including inflow chamber 52 and outflow chamber 54. Flow regulator 12 may also be provided with power source 45 (such as a motor) which can be coupled to one or both of pumping members 44 and 46 (such as through a drive shaft, belt, or other suitable connection mechanism) to provide active driving of the pumping members. Power source 45 is preferably a battery powered motor housed on or within housing 42 or is a remotely actuable motor, actuable by an actuator external to the body through wireless or wired connection. Such an actuator may, for example, be a power source for energizing motor 45 and selectively connectable to motor 45 through an operator actuable switch with electrical connection being made to motor 45 with electrical conductors extending within the vasculature through a suitable catheter.

Pumping member 44 has a plurality of centrifugal fins 56 which are mounted to a first side of a generally circular base plate 58. A gear 60 having teeth 62 is mounted to a second side of base plate 58. Only two fins 56 are shown in FIG. 2. However, pumping member 44 preferably has three or more fins, as described later in the specification. Pumping member 44 is configured to rotate, orthogonal to the plane of the paper of FIG. 2, generally about an axis of rotation 62. Fins 56 are coupled to a thrust bearing 64 which is nested in a thrust bearing seat 66 on the wall of housing 42. Pumping member 44 spins about axis 62 with thrust bearing 64 bearing against thrust bearing seat 66.

Similarly, pumping member 46 is provided with a plurality of fins 68. As with pumping member 44, only two fins 68 are shown in FIG. 2, but pumping member 46 preferably has three or more fins. As with pumping member 44, fins 68 of pumping member 46 are disposed on a first side of a generally circular base plate 70, while a gear 72 having gear teeth 74 is disposed on the opposite side of base plate 70. Pumping member 46 also has a thrust bearing 74 which sits in a thrust bearing seat 76 in housing 42. Pumping member 46 is configured to rotate also orthogonally to the page of FIG. 2, about an axis of rotation 78 wherein thrust bearing 74 bears against thrust bearing seat 76 to accommodate such rotation.

The gear teeth 74 of gear 72 are engaged with the gear teeth 62 of gear 60 on pumping member 44. As illustrated in FIG. 2, gear 72 is smaller than gear 60. Thus, through the gear ratios applied by gears 72 and 60, pumping member 44 rotates at a slower speed than pumping member 46.

In operation, blood flow is generally indicated by arrow 80. Blood flows from heart 16 in through inflow end 34 into inflow chamber 52 of regulator 12. The blood encounters the fins 68 on pumping member 46 and causes pumping member 46 to rotate.

The blood flow, through rotation of fins 68 on pumping member 46, is brought into an internal chamber between walls 48 and 50. The blood flow then crosses over to pumping member 44. Rotation of pumping member 44 causes the blood to exit to outflow chamber 54, and eventually out through outflow end 36.

Since gear 72 is smaller than gear 60, pumping member 46 spins faster than pumping member 44.

Therefore, blood enters through inflow chamber 52 at a first velocity and causes pumping member 46 to spin. However, the blood exits through outflow chamber 54 at a slower rate, because of the gear ratio applied by gears 60 and 72, which causes pumping member 44 to spin at a slower rate than pumping member 46. This operates to step down the velocity of the blood flowing through regulator 12. This results in reduced blood velocity reaching ascending aorta 18 and baroreceptors 30 and 32.

FIG. 3 is a side view of pumping member 46. It will be appreciated that pumping member 44 is similar to pumping member 46 (other than the difference in gear sizes) and therefore only pumping member 46 is described in detail. FIG. 3 illustrates that fins 68 are preferably curved to accomplish more efficient pumping of blood. FIG. 3 also illustrates that, in one preferred embodiment, fins 68 terminate in a center region of pumping member 46 at thrust bearing 74. Gear 72 is also shown in phantom in FIG. 3.

FIG. 4 is a side view of pumping member 46. FIG. 4 illustrates that the fins are preferably curved downwardly at a radial outward portion 82 thereof. This is to accommodate the curved shape of housing 42.

FIG. 5 is another view of pumping member 46 showing the opposite side of that shown in FIG. 3. FIG. 5 also illustrates that gear 72 is preferably concentrically arranged about the axis of rotation 78.

FIG. 6 is end view looking into flow regulator 12 from inflow end 34. FIG. 6 illustrates that wall member 50 is preferably hemispherical in shape and covers approximately half of the internal width of housing 42, in order to better define inflow chamber 52.

Flow regulator 14 is preferably substantially identical to flow regulator 12, except that it is

rotated 180° within descending aorta 20. Thus, outflow end 36 of flow regulator 12 corresponds to inflow end 38 of flow regulator 14, and inflow end 34 of flow regulator 12 corresponds to outflow end 40 of flow regulator 14. Of course, blood flow through flow regulator 14 is in an opposite direction to that shown by arrow 80 in FIG. 2.

Flow regulator 14 acts to increase (or step up) blood flow velocity to renal arteries 26 and 28. This utilizes the physiological feedback response discussed above to reduce stress on the heart.

Active control of regulators 12 and 14 can be accomplished with the above-mentioned power source or motor 45. Such control can be obtained by synchronizing it to the sinus rhythms in much the same way as that described with respect to FIG. 8 below. Also, regulators 12 and 14 can be separately controlled to control blood flow and pressure to the baroreceptors and renal arteries separately. Also, the control pulses can be shaped so that flow and pressure are controlled smoothly rather than abruptly. Further, the geometry of the elements in regulators 12 and 14 can be changed to accomplish desired changes in flow characteristics therethrough. For instance, changing the diameter of the inflow and outflow ends changes the velocity of fluid flowing therethrough. Also, changing the shape of gears 60 and 72 (such as making them elliptical) changes the rotational characteristics of the pumping members 44 and 46. Further, either one or both of regulators 12 and 14 can be used.

FIG. 7A illustrates a second embodiment of a system 90 for treating heart disease in accordance with another aspect of the present invention. System 90 includes balloons 94 and 96 (described below) and is

shown disposed in descending aorta 20 proximate renal arteries 26 and 28. FIG. 7B shows that system 90 can include a second portion (with balloons 94' and 96') disposed proximate baroreceptors 30 and 32. The second portion operates similarly to the first portion described below, but the second portion acts to selectively increase or decrease flow to baroreceptors 30 and 32 as will be appreciated and will not be described in great detail for the sake of simplicity.

System 90 (as shown in FIG. 7A) includes a catheter 92 which has, at its proximal end, a first expansion member 94 and a second expansion member 96. System 90 also includes inflation controller 98 and heart rate monitor 100. Catheter 92 is preferably a multi-lumen catheter such that expandable members 94 and 96 (and 94' and 96') are expandable independently of one another. Thus, inflation controller 98 is preferably a pneumatic inflation device which has a pair of pneumatic outputs 102 and 104 (and two additional pneumatic outputs 102' and 104' for connection to balloons 94' and 96') which are connected to the lumens of catheter 92 which are, in turn, connected to inflation members 94 and 96 (as is described in greater detail with respect to FIG. 10). In operation, balloons 94 and 96 are inflated and deflated to increase blood flow to the renal arteries 26 and 28 in order to inhibit the renin-angiotensin response, and thus prevent fluid volume retention.

The operation of system 90 is described with respect to FIGS. 8 and 9A-9D. FIG. 8 illustrates a sinus rhythm 106 and a corresponding timing diagram 108. Only a portion of system 90 is shown in FIGS. 9A-9D for the sake of clarity.

Initially, catheter 92, with balloons 94 and 96, is introduced into the vascular system, such as through a femoral artery. Catheter 92 is positioned such that balloons 94 and 96 are placed across the renal arteries 26 and 28 as illustrated in FIG. 9A, with both balloons deflated. At systole, balloon 96 remains deflated while balloon 94 is inflated. This is shown in FIG. 9B, and causes a pool of blood 110 to be accumulated in the descending aorta in the region across renal arteries 26 and 28.

After a finite delay, balloon 96 is inflated as shown in FIG. 9C. As balloon 96 is inflated, the blood pressure between the balloons begins to increase, and blood is forced into renal arteries 26 and 28 as illustrated by arrows 112 and 114.

After balloon 96 has been inflated, or while balloon 96 is being inflated, balloon 94 is deflated as indicated in FIG. 9D. This functions to prevent excessive pressure and flow from being exerted on renal arteries 26 and 28. This allows the blood which had accumulated in the area of renal arteries 26 and 28 to escape from that region and continue flowing through the rest of the vasculature.

It should be noted that the entire inflation and deflation sequence shown in FIGS. 9A-9B takes place preferably in a time period less than one heart beat so that, at the next systole, the sequence can be repeated. The balloon inflation and deflation times, as well as the pressures and the inflation and deflation sequences, are gated to the patient's heartbeat through external heart rate monitoring equipment, such as heart rate monitor 100.

The increased blood flow to the renal system inhibits the renin-angiotensin system response and thus

reduces the likelihood that any fluid volume retention will occur. Since little or no excess fluid volume is accumulated, there is a smaller load on heart 16. This allows heart 16 to passively recuperate, or it renders pharmacologically supplemented recuperation more efficient.

FIG. 10 is a more detailed view of catheter 92. In a preferred embodiment, catheter 92 includes a proximal hub 116 with a pair of proximal coupling members 118 and 120. Coupling members 118 and 120 are preferably coupled to a pair of lumens 122 and 124 within the body of catheter 92. Lumen 122 extends at least to balloon 94 and has an aperture 126 formed therein which fluidly communicates with balloon 94. Lumen 124 extends at least to balloon 96 and has an aperture 128 which fluidly communicates with the interior of balloon 96. Thus, as inflation controller 98 provides pneumatic pressure within lumens 122 and 124, balloons 94 and 96 can be inflated and deflated, as desired.

As described above, system 90 can be arranged proximate the renal system or the baroreceptors or both. Thus, blood flow in those areas can be controlled in a synchronous fashion, or entirely independently of one another.

Thus, it can be seen that the present invention provides a flow regulation system for the treatment of congestive heart failure. The flow regulation system regulates flow to use the bodies neural and physiological feedback systems to control the heart to relieve the heart of congestion. The present system also provides the ability to controllably allow the heart to recuperate and increase the heart rate to a normal level and thus allow for increased, normal

cardiac output. This allows the heart to recuperate, and increases the efficiency of pharmacologically supplemented recuperation methods.

Although the present invention has been described with reference to preferred embodiments, workers skilled in the art will recognize that changes may be made in form and detail without departing from the spirit and scope of the invention.

WHAT IS CLAIMED IS:

1. A treatment system for treating heart disease in a mammal having a coronary system including a heart, an ascending aorta, baroreceptors, a descending aorta, a renal system including renal arteries, and a physiological feedback system controlling the heart, the treatment system comprising:

a regulator disposed in the coronary system and configured to regulate at least one of blood pressure and blood flow through the coronary system to affect the physiological feedback system so as to reduce heart congestion.

2. The treatment system of claim 1 wherein the flow regulator comprises:

a first flow regulator disposed in the coronary system to regulate blood flow through the coronary system to one of the renal arteries and the baroreceptors.

3. The treatment system of claim 2 wherein the first flow regulator is disposed in the descending aorta proximate the renal arteries to regulate blood flow velocity to the renal arteries from the descending aorta.

4. The treatment system of claim 2 wherein the first flow regulator is disposed in the ascending aorta proximate the baroreceptors to regulate blood flow velocity to the baroreceptors from the heart.

5. The treatment system of claim 1 wherein the regulator comprises:

a first regulator disposed in the coronary system to regulate blood pressure in the coronary system.

6. The treatment system of claim 5 wherein the first regulator is disposed in the descending aorta proximate the renal arteries to regulate blood pressure in the renal arteries.

7. The treatment system of claim 5 wherein the first regulator is disposed in the ascending aorta proximate the baroreceptors to regulate blood pressure at the baroreceptors.

8. The treatment system of claim 1 wherein the regulator comprises a first regulator disposed in the descending aorta proximate the renal arteries and including:

a first expandable member;

a second expandable member; and

an expander coupled to the first expandable member and the second expandable member and configured to controllably expand the first and second expandable members.

9. The treatment system of claim 8 wherein the first expandable member is disposed in the descending aorta upstream of the renal arteries, the second expandable member is disposed in the descending aorta down stream of the renal arteries, and the first and second expandable members are expandable to have a dimension sufficient to substantially occlude the descending aorta.

10. The treatment system of claim 9 wherein the expander is configured to control expansion of the first and second expandable members to periodically increase blood flow to the renal arteries above naturally occurring blood flow.

11. The treatment system of claim 9 wherein the expander is configured to contract the first expandable

member and expand the second expandable member to allow blood to pool in the descending aorta proximate the renal arteries and to then expand the first expandable member to increase blood pressure and blood flow into the renal arteries.

12. The treatment system of claim 11 wherein the expander is configured to contract the second expandable member after the first expandable member has been expanded.

13. The treatment system of claim 11 wherein the expander is configured to contract the second expandable member as the first expandable member is being expanded.

14. The treatment system of claim 11 wherein the expander is configured to control expansion of the first and second expandable members based on a sinus rhythm of the heart.

15. The treatment system of claim 14 and further comprising:

a heart rate monitor coupled to the expander and providing a heart rate signal indicative of heart rate, and wherein the expander is configured to control expansion of the first and second expandable members based on the sinus rhythm indicated by the heart rate signal.

16. The treatment system of claim 8 wherein the first and second expandable members are balloons and further comprising:

a catheter coupled to the first and second expandable members and having a proximal end and a distal end and first and second lumens therein, the first lumen being in fluid communication with the

first expandable member and the second lumen being in fluid communication with the second expandable member, the catheter including proximal couplers configured to be coupled to the expander to receive inflation fluid therefrom.

17. The treatment system of claim 8 wherein the regulator further comprises:

a second regulator disposed in the ascending aorta and configured to regulate flow through the ascending aorta.

18. The treatment system of claim 17 wherein the second regulator comprises:

a third expandable member; and

a fourth expandable member, wherein the expander is coupled to the third expandable member and the fourth expandable member and is configured to controllably expand the third and fourth expandable members.

19. The treatment system of claim 18 wherein the third expandable member is disposed in the ascending aorta upstream of the baroreceptors, the fourth expandable member is disposed downstream of the baroreceptors, and the third and fourth expandable members are expandable to have a dimension sufficient to substantially occlude the ascending aorta.

20. The treatment system of claim 19 wherein the expander is configured to control expansion of the third and fourth expandable members to periodically increase and decrease blood flow to the baroreceptors above and below naturally occurring blood flow, respectively.

21. The treatment system of claim 20 wherein the expander is configured to expand the first, second,

third and fourth expandable members independently of one another.

22. The treatment system of claim 17 wherein the first regulator comprises a step-up pump having an inlet end and an outlet end, the inlet end receiving blood flow at a first rate, the step-up pump being configured to provide blood flow through the outflow end at a second rate, slower than the first rate.

23. The treatment system of claim 22 wherein the second regulator comprises:

a step-down pump having an inlet end and an outlet end, the inlet end receiving blood flow at a first rate, the step-down pump being configured to provide blood flow through the outflow end at a second rate, faster than the first rate.

24. The treatment system of claim 23 wherein the step-up pump and the step-down pump each include:

a housing;

a first rotatable pumping member with a gear coupled thereto rotatably disposed in the housing; and

a second rotatable pumping member with a gear coupled thereto rotatably disposed in the housing, the gears on the first and second pumping members being of different size and being engageable with one another such that rotation of the first pumping member at a first rate causes rotation of the second pumping member at a second rate, different from the first rate.

25. The treatment system of claim 24 wherein the first and second pumping members each include a plurality of fins coupled to the gear.

26. The treatment system of claim 23 and further comprising a motor, coupled to the step-up and step-down pumps to actively drive the step-up and step-down pumps.

27. A method of treating heart disease in a mammal having a coronary system including a heart, an ascending aorta, baroreceptors, and a descending aorta, a renal system including renal arteries, and a physiological feedback system controlling the heart, the treatment system comprising:

providing a regulator in the coronary system; and

regulating flow of blood in the coronary system to affect the physiological feedback system such that heart congestion is reduced.

28. The method of claim 27 wherein providing a regulator comprises:

providing a first regulator disposed in the ascending aorta proximate the renal arteries to regulate flow of blood to the renal arteries from the descending aorta.

29. The method of claim 28 wherein providing the first regulator comprises:

providing a first expandable member;

providing a second expandable member; and

disposing the first expandable member in the descending aorta upstream of the renal arteries; and

disposing the second expandable member in the ascending aorta downstream of the renal

arteries, wherein the first and second expandable members are expandable to have a dimension sufficient to substantially occlude the descending aorta.

30. The method of claim 29 and further comprising:
controlling expansion of the first and second expandable members to periodically increase blood flow to the renal arteries in excess of naturally occurring blood flow.

31. The method of claim 30 wherein expanding the first and second expandable members comprises:
contracting the first expandable member;
expanding the second expandable member to allow blood to pool in the descending aorta proximate the renal arteries; and
then expanding the first expandable member to increase blood pressure and blood flow into the renal arteries.

32. The method of claim 31 and further comprising:
contracting the second expandable member after the first expandable member has been expanded.

33. The method of claim 31 and further comprising:
contracting the second expandable member as the first expandable member is being expanded.

34. The method of claim 31 wherein expanding the first expandable member and expanding the second expandable member comprises:
controlling expansion of the first and second expandable members based on a sinus rhythm of the heart.

35. The method of claim 28 and further comprising:
providing a second regulator disposed in the ascending aorta; and
regulating flow of blood through the ascending aorta with the second regulator.

36. The method of claim 35 wherein regulating flow of blood with the first regulator comprises increasing blood flow velocity to the renal arteries.

37. The method of claim 35 wherein regulating flow with the second regulator comprises:
decreasing flow velocity to the renal system with the second flow regulator.

38. A method of treating heart disease in a mammal having a coronary system including a heart beating at a heart rate, an ascending aorta, baroreceptors, a descending aorta, a renal system including renal arteries, and a physiological feedback system controlling the heart, the method comprising:
placing a regulator in the coronary system to regulate flow of blood in the coronary system; and
regulating the flow of blood in the coronary system by actuating the regulator, within a periodicity of the heart rate to selectively increase and decrease blood flow in a portion of the coronary system to affect the physiological feedback system so as to reduce heart congestion.

39. The method of claim 38 wherein placing a regulator comprises:

providing a regulator in the ascending aorta, proximate the baroreceptors, to control blood flow to the baroreceptors.

40. The method of claim 39 wherein regulating comprises:

controlling blood flow to the baroreceptors to decrease blood flow to the baroreceptors below naturally occurring blood flow.

41. The method of claim 39 wherein regulating comprises:

selectively decreasing blood pressure at the baroreceptors within the periodicity of the heart rate, below naturally occurring blood pressure.

42. The method of claim 38 wherein placing a regulator comprises:

providing a regulator in the descending aorta, proximate the renal system, to control blood flow to the renal system.

43. The method of claim 42 wherein regulating comprises:

selectively increasing and decreasing blood flow to the renal system to periodically increase blood flow in the renal system above naturally occurring blood flow.

44. The method of claim 42 wherein regulating comprises:

selectively increasing and decreasing blood flow to the renal system to periodically increase blood pressure in the renal system above naturally occurring blood pressure.

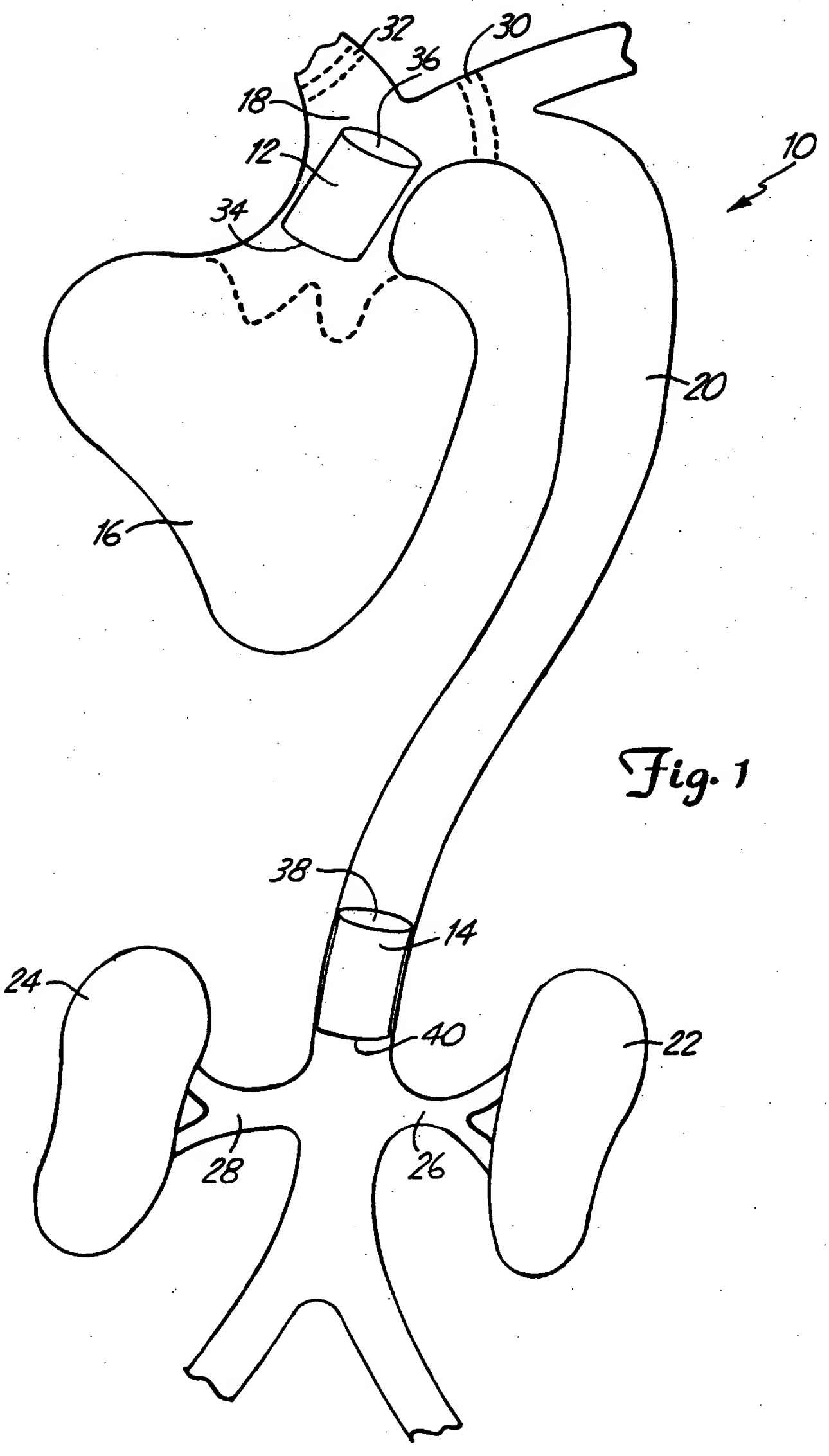


Fig. 1

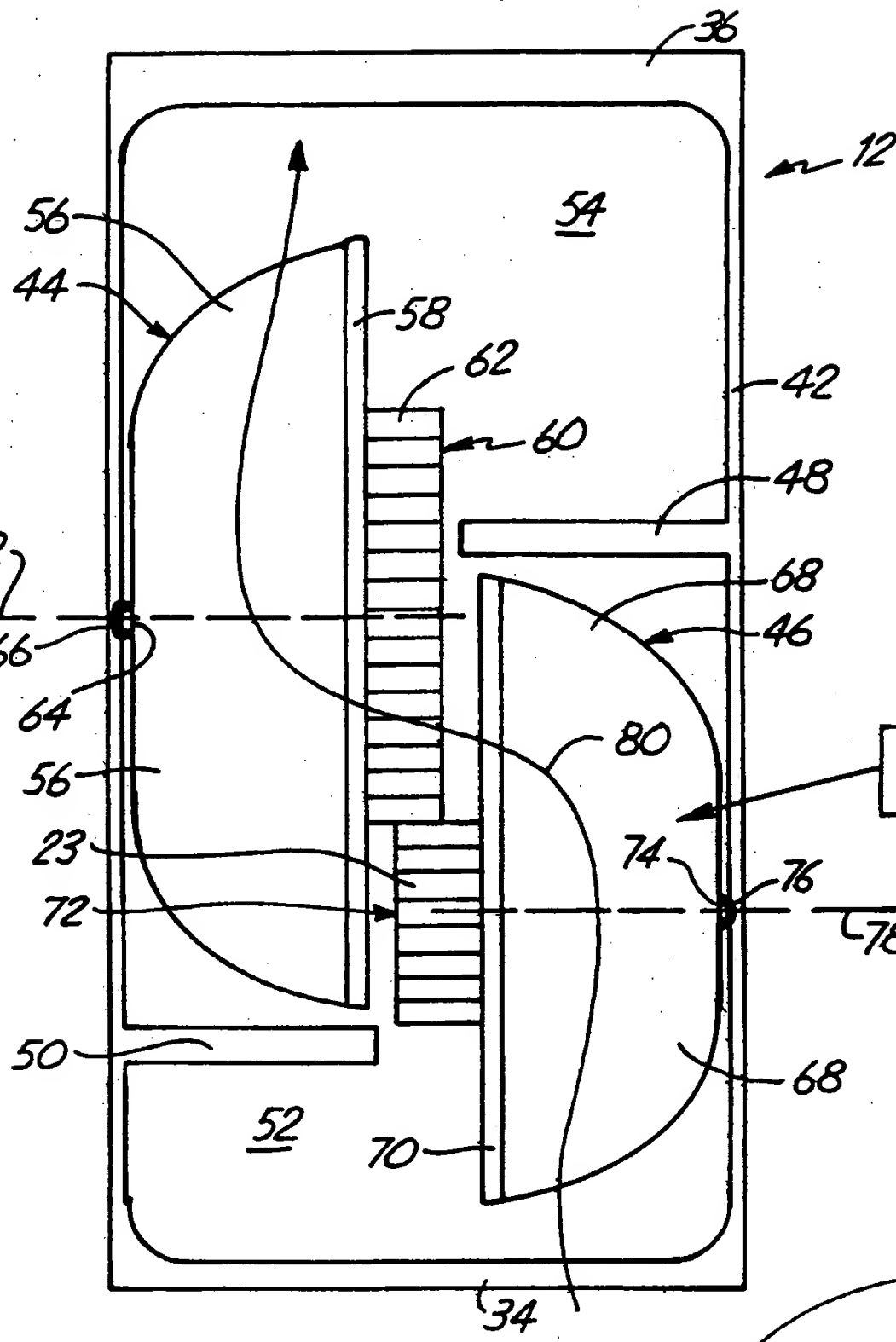


Fig. 2

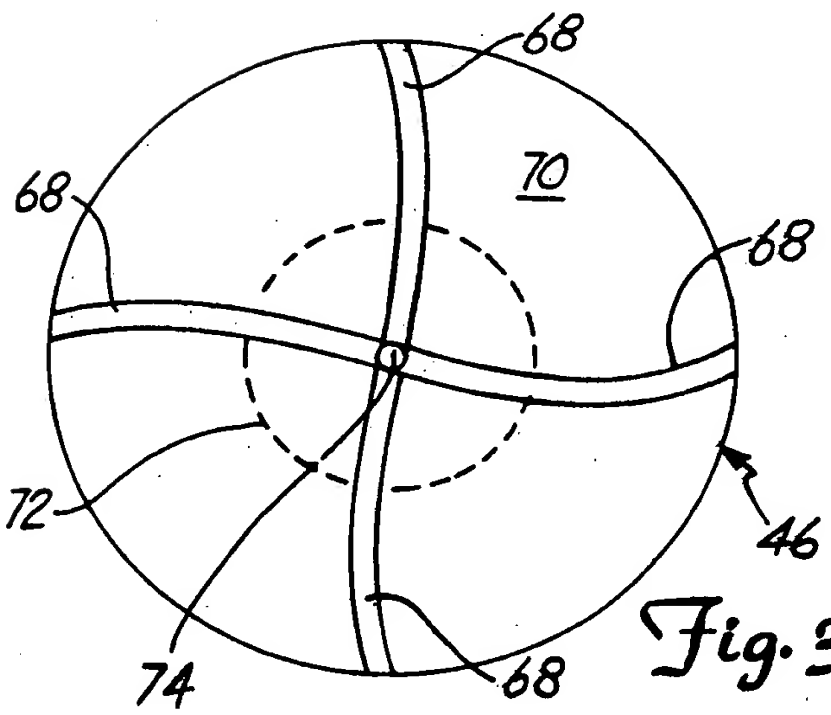


Fig. 3

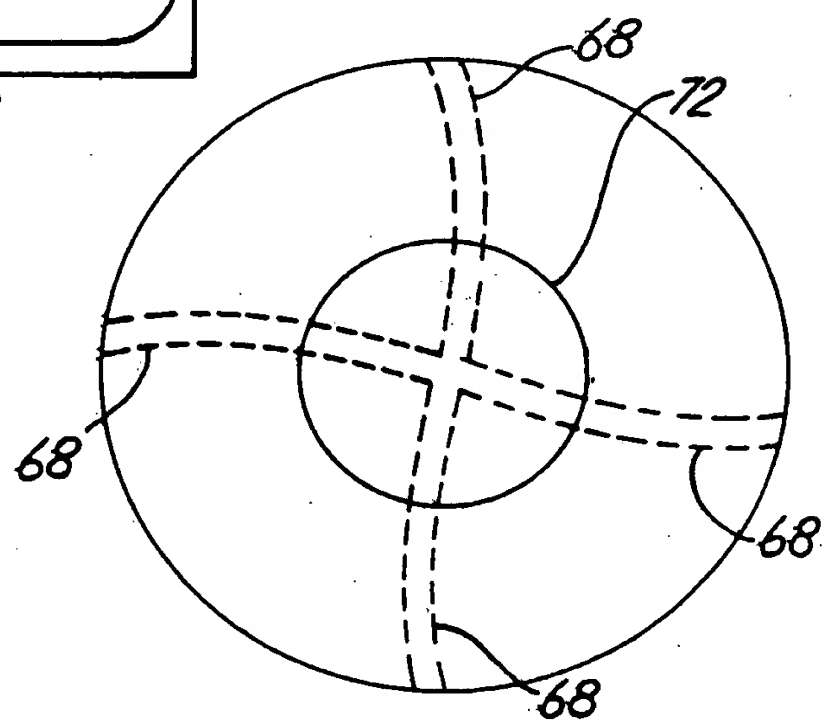


Fig. 5

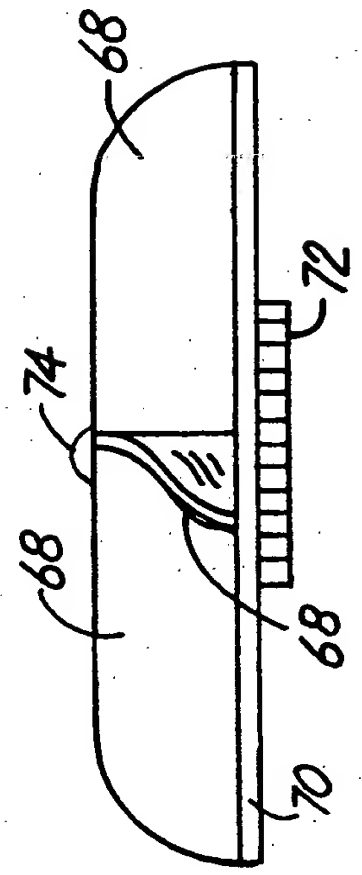


Fig 4

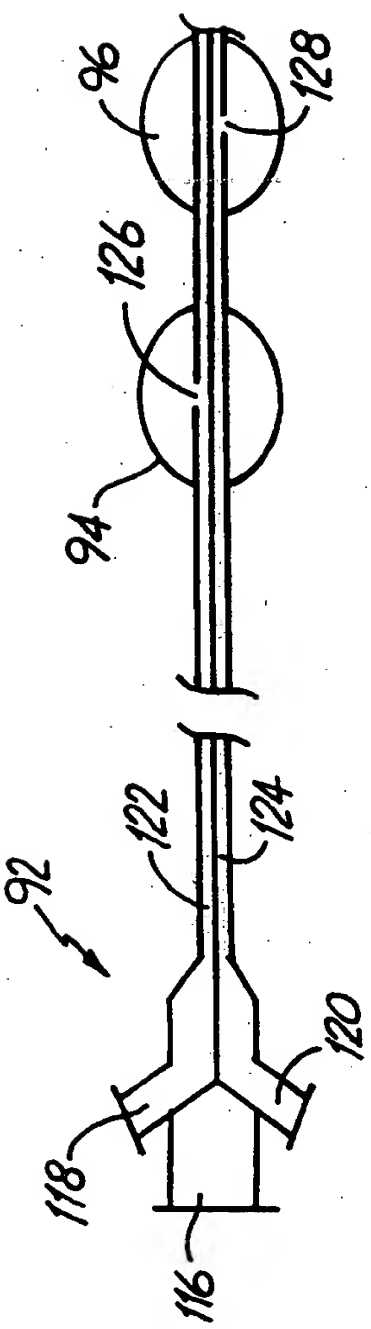


Fig 10

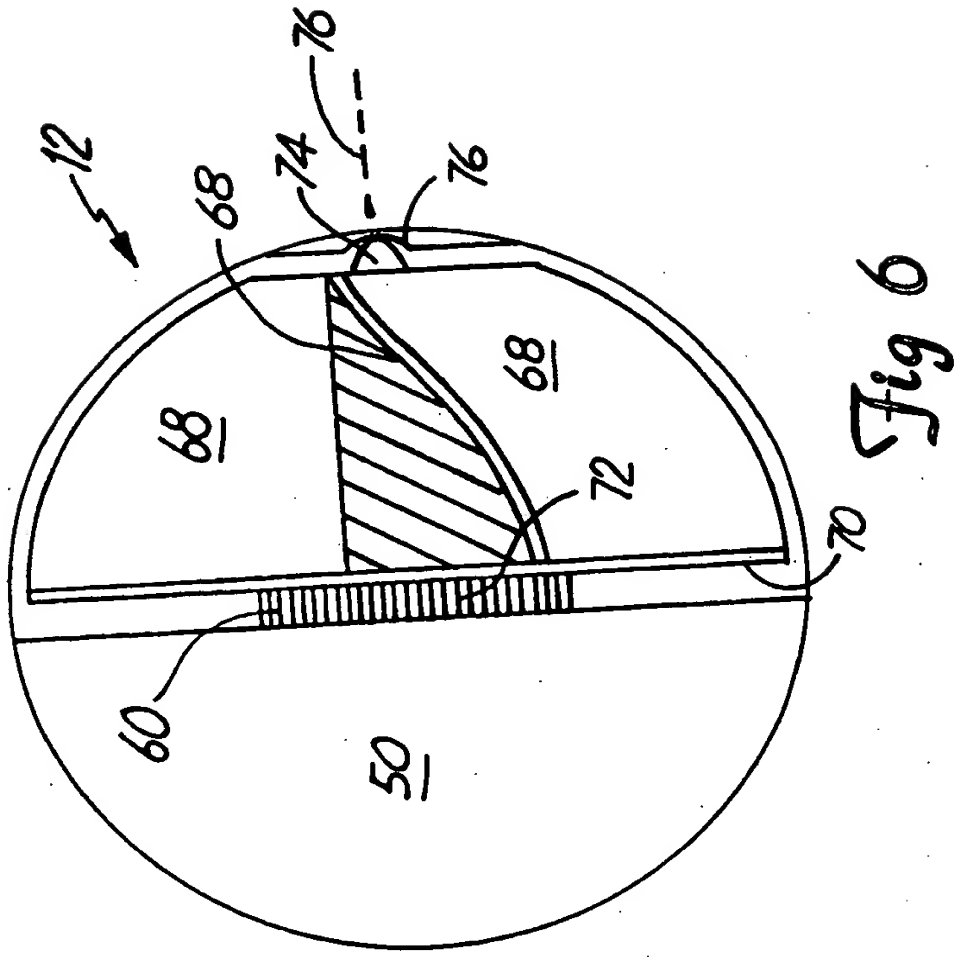


Fig 6

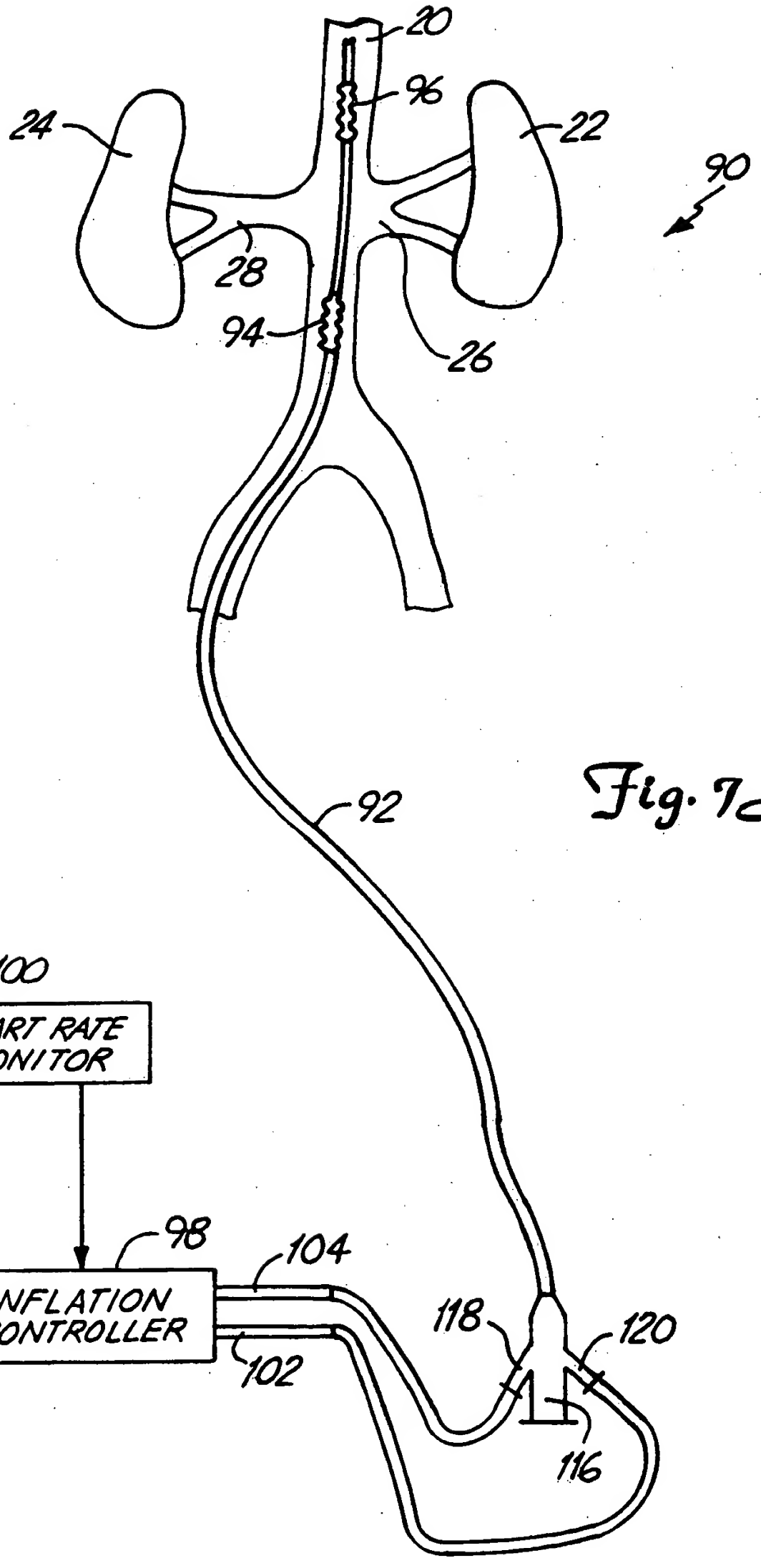


Fig. 7A

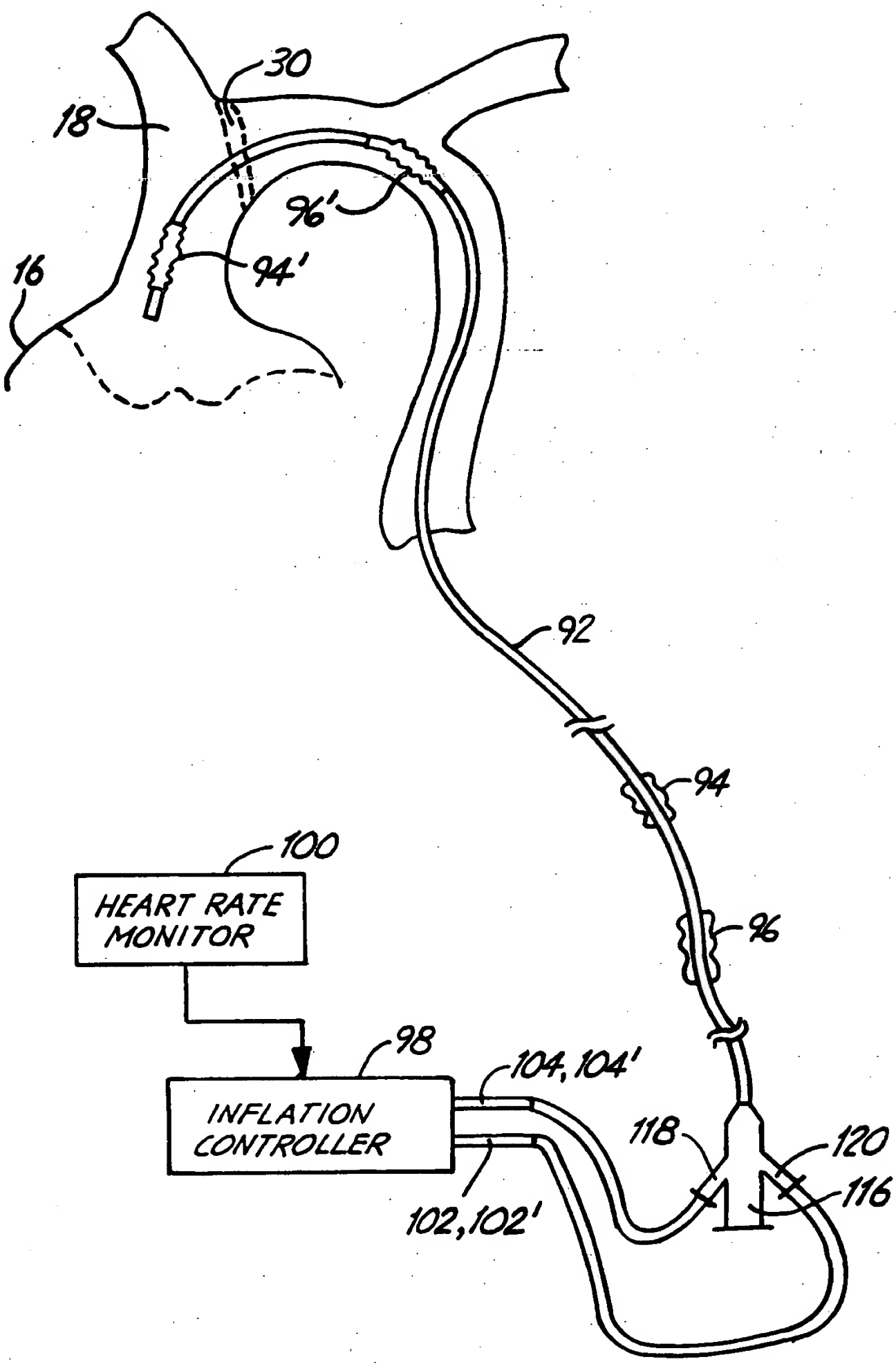


Fig. 7B

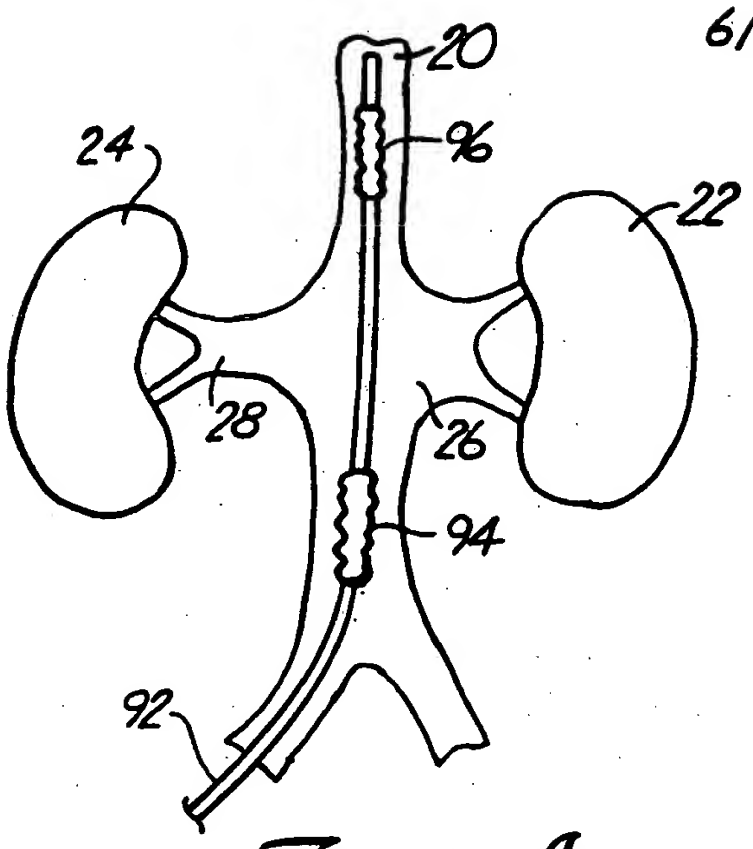


Fig. 9A

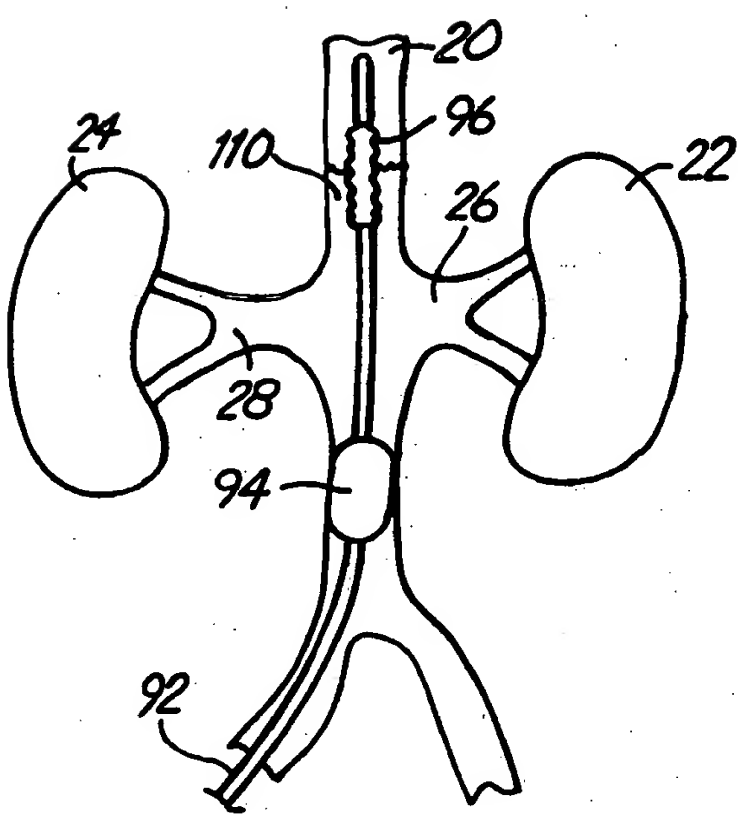


Fig. 9B

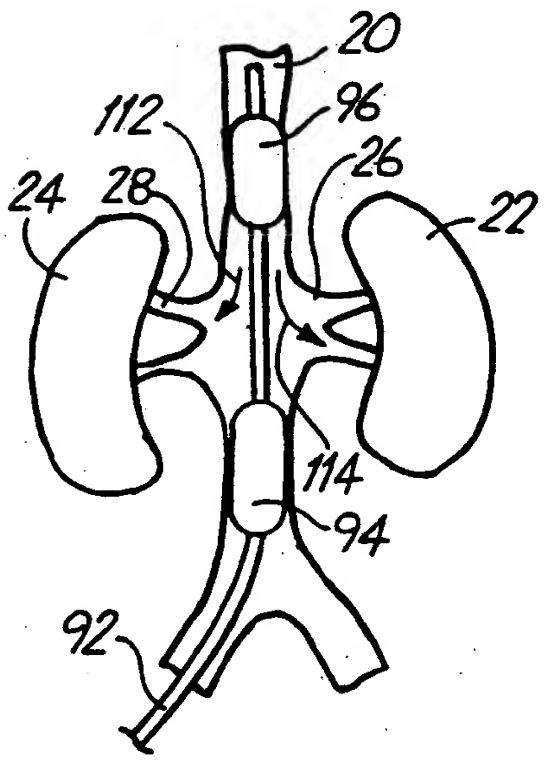


Fig. 9C

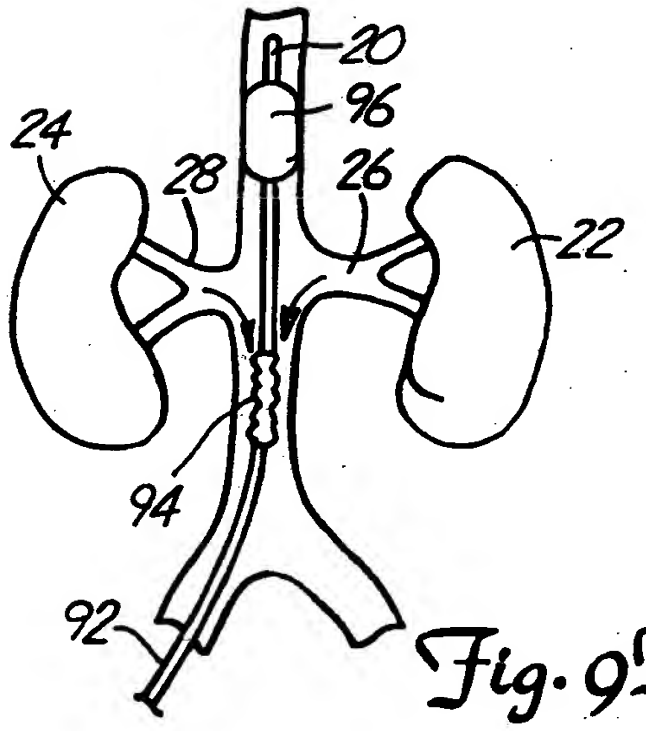


Fig. 9D

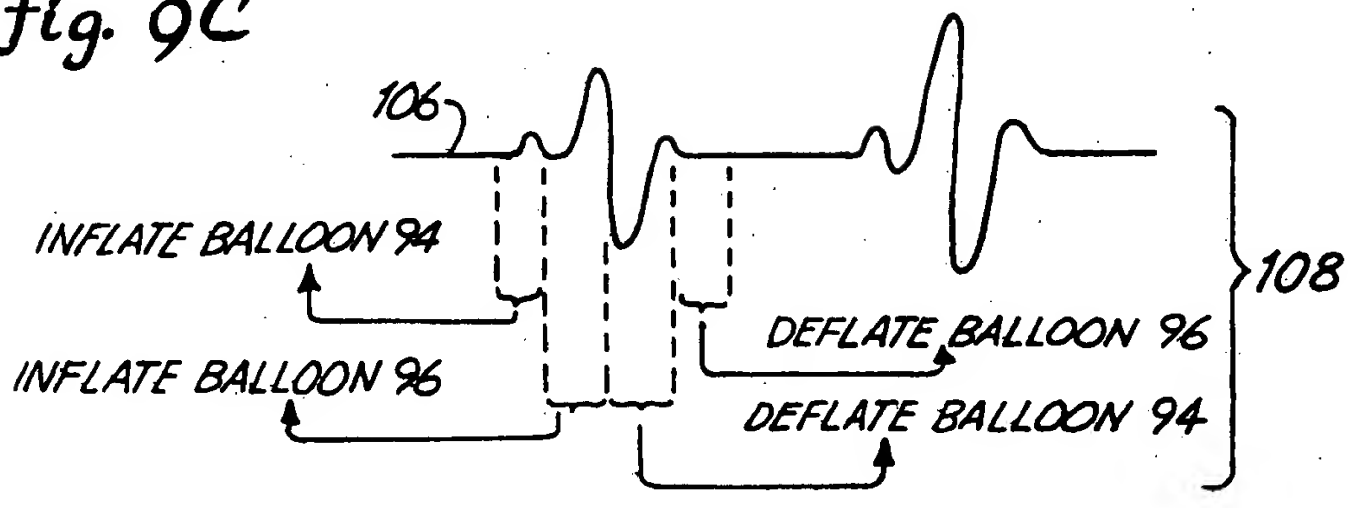


Fig. 8

INTERNATIONAL SEARCH REPORT

Int. donal Application No
PCT/US 99/07411

A. CLASSIFICATION OF SUBJECT MATTER
IPC 6 A61M1/10 A61M25/10

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)
IPC 6 A61M

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic data base consulted during the international search (name of data base and, where practical, search terms used)

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category °	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
X	US 4 685 446 A (CHOY) 11 August 1987 (1987-08-11) column 1, line 5 - line 26 column 1, line 64 - column 2, line 51 ---	1
X	US 4 964 864 A (SUMMERS ET AL.) 23 October 1990 (1990-10-23) column 1, line 5 - line 33 column 2, line 23 - line 34 column 4, line 8 - line 47 figures 1,3,6 ---	1,2,4,5, 7
A	EP 0 654 283 A (ANAYA FERNANDEZ DE LOMANA) 24 May 1995 (1995-05-24) column 2, line 27 - line 37 column 4, line 31 - line 58 column 7, line 3 - line 27 figures 1,3,4 --- -/--	8-10,15, 16

[X] Further documents are listed in the continuation of box C.

[X] Patent family members are listed in annex.

° Special categories of cited documents :

"A" document defining the general state of the art which is not considered to be of particular relevance

"E" earlier document but published on or after the international filing date

"L" document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)

"O" document referring to an oral disclosure, use, exhibition or other means

"P" document published prior to the international filing date but later than the priority date claimed

"T" later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention

"X" document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone

"Y" document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art.

"&" document member of the same patent family

Date of the actual completion of the international search	Date of mailing of the international search report
26 July 1999	02/08/1999

Name and mailing address of the ISA	Authorized officer
European Patent Office, P.B. 5818 Patentlaan 2 NL - 2280 HV Rijswijk Tel. (+31-70) 340-2040, Tx. 31 651 epo nl, Fax: (+31-70) 340-3016	Schönleben, J

1

Form PCT/ISA/210 (second sheet) (July 1992)

INTERNATIONAL SEARCH REPORT

Int. tional Application No
PCT/US 99/07411

C.(Continuation) DOCUMENTS CONSIDERED TO BE RELEVANT

Category *	Citation of document, with indication,where appropriate, of the relevant passages	Relevant to claim No.
A	US 5 308 320 A (SAFAR ET. AL.) 3 May 1994 (1994-05-03) column 8, line 16 - line 32 figure 5	17-19

1

Form PCT/ISA/210 (continuation of second sheet) (July 1992)

INTERNATIONAL SEARCH REPORT

...ernational application No.
PCT/US 99/07411

Box I Observations where certain claims were found unsearchable (Continuation of item 1 of first sheet)

This International Search Report has not been established in respect of certain claims under Article 17(2)(a) for the following reasons:

1. [X] Claims Nos.: 27-44
because they relate to subject matter not required to be searched by this Authority, namely:
Rule 39.1(iv) PCT - Method for treatment of the human or animal body by therapy and/or surgery.

2. [ ] Claims Nos.:
because they relate to parts of the International Application that do not comply with the prescribed requirements to such an extent that no meaningful International Search can be carried out, specifically:

3. [ ] Claims Nos.:
because they are dependent claims and are not drafted in accordance with the second and third sentences of Rule 6.4(a).

Box II Observations where unity of invention is lacking (Continuation of item 2 of first sheet)

This International Searching Authority found multiple inventions in this international application, as follows:

1. [ ] As all required additional search fees were timely paid by the applicant, this International Search Report covers all searchable claims.

2. [ ] As all searchable claims could be searched without effort justifying an additional fee, this Authority did not invite payment of any additional fee.

3. [ ] As only some of the required additional search fees were timely paid by the applicant, this International Search Report covers only those claims for which fees were paid, specifically claims Nos.:

4. [ ] No required additional search fees were timely paid by the applicant. Consequently, this International Search Report is restricted to the invention first mentioned in the claims; it is covered by claims Nos.:

Remark on Protest

[ ] The additional search fees were accompanied by the applicant's protest.

[ ] No protest accompanied the payment of additional search fees.

INTERNATIONAL SEARCH REPORT

Information on patent family members

Int .tional Application No
PCT/US 99/07411

Patent document cited in search report	Publication date	Patent family member(s)	Publication date
US 4685446 A	11-08-1987	US 4902273 A	20-02-1990
		US 4771765 A	20-09-1988
US 4964864 A	23-10-1990	AU 641430 B	23-09-1993
		AU 6369990 A	11-06-1992
		EP 0480101 A	15-04-1992
		US 5112349 A	12-05-1992
EP 654283 A	24-05-1995	ES 2077519 A	16-11-1995
		AT 178805 T	15-04-1999
		CA 2136407 A	23-05-1995
		DE 69417847 D	20-05-1999
		JP 7255836 A	09-10-1995
		US 5505701 A	09-04-1996
US 5308320 A	03-05-1994	US 5820593 A	13-10-1998
		US 5879316 A	09-03-1999
		US 5906588 A	25-05-1999
		US 5383854 A	24-01-1995